

SCIENTIFIC LETTER

Acute effects of 40% oxygen supplementation in adults with cyanotic congenital heart disease

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This study examined the acute effects of 40% oxygen supplementation in adults with cyanotic congenital heart disease (CCHD). These young patients have significant morbidity and premature mortality, relating in part to their cyanotic state. We have shown that systemic arterial oxygen saturation (SAOS) increases significantly when these patients are administered 40% oxygen, irrespective of their underlying congenital cardiovascular diagnosis or the presence of pulmonary hypertension. Forty per cent oxygen supplementation increased SAOS above 85% in the majority of patients, with none having an SAOS less than 80%. This improvement in saturation may be clinically important because significant erythrocytosis is unusual if SAOS is greater than 85% and it is this physiological adaptation which is responsible for many of the symptoms experienced by these patients.

About 10% of adults with congenital heart disease are cyanosed and although their functional capacity is good up to the third decade, it declines along with quality of life thereafter when complications and symptoms related to the cyanotic state become more frequent. Cyanosis occurs when hypoxaemia stimulates erythropoiesis causing erythrocytosis. This re-establishes tissue oxygen delivery at the expense of an elevated haematocrit and leads to symptoms including, headaches, impaired mentation, lassitude, and myalgia, all of which have an impact on quality of life and physical ability.¹ In addition to these hyperviscosity symptoms, there is a direct relation between SAOS and exercise capacity in these patients and a low SAOS is a recognised risk factor for death.²

The fundamental physiological fault in CCHD is the diversion of systemic venous blood into the systemic arterial circulation, therefore SAOS is determined by the amount of pulmonary blood flow and the degree of right to left shunting. It is generally believed that pulmonary vascular reactivity in adults with CCHD is negligible and therefore the use of pulmonary vasodilators, such as supplemental oxygen to increase oxygen saturation, is not advocated. In the absence of evidence to support oxygen therapy it is felt the risk of mucosal dehydration and epistaxis outweighs any benefits. Patients who use home oxygen (25–40%) however, do report an improvement in symptoms. We therefore examined the acute effects of 40% oxygen supplementation on SAOS in 29 adults with CCHD.

METHODS

Seventy five adult patients with CCHD were identified from the adult congenital cardiac database of the Toronto General Hospital. Thirty patients agreed to participate and 29 were eligible for inclusion having a resting SAOS \leq 90% and no evidence or history of respiratory disease or sleep apnoea (mean age 37 (10.7), range 20–63 years). Seventeen patients had complex palliated congenital heart disease and 12 patients had simple defects with Eisenmenger physiology (defined as those with pulmonary hypertension and reversed

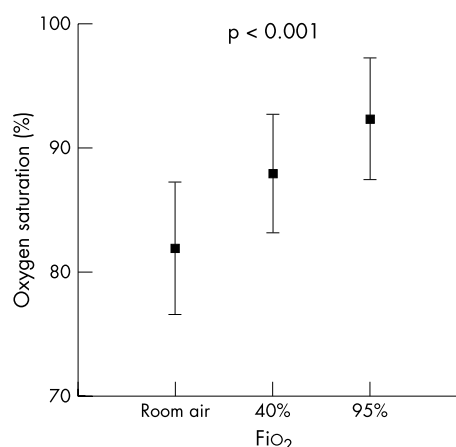


Figure 1 Change in systemic arterial oxygen saturation (SAOS) with administration of 40% and 95% oxygen (FiO₂).

or bidirectional shunting at ventricular, atrial or great artery level). SAOS was measured using a digital pulse oximeter (Nellor N20, Pleasanton, California, USA). Blood pressure and heart rate were also recorded. Forty per cent supplemental oxygen, followed by 95% oxygen was administered, each for 10 minutes inhalation duration.

RESULTS

Data are presented as mean (SD). Differences in arterial oxygen saturation were analysed using analysis of variance (ANOVA) for repeated measures. Relations between the baseline variables and response to supplemental oxygen were assessed using Pearson correlation coefficient. Changes were considered significant if $p < 0.05$.

In room air, mean SAOS was 81.6 (5.4%), range 73–90%, mean haemoglobin was 185 (27.5) g/l, range 127–247 g/l, and mean haematocrit was 0.56 (0.09) l/l, range 0.37–0.78 l/l. There was a significant association between haematocrit and baseline SAOS ($r = -0.46$, $p = 0.01$). Apart from pulmonary artery pressure (higher in Eisenmenger patients), there were no other significant differences in baseline characteristics between Eisenmenger and non-Eisenmenger patients: haemoglobin concentration (187 (22) g/l v 184 (30) g/l, $p > 0.1$); haematocrit (0.57 (0.08) v 0.57 (0.10), $p > 0.1$); and resting SAOS (82.5 (6.4)% v 81.7 (4.9)%, $p > 0.01$). Oxygen supplementation increased SAOS in all subjects, from 81.6 (5.4)% in room air to 88.0 (4.8)% (range of increase 2–11%) while breathing 40% oxygen, to 92.4 (4.9)% (range of increase 5–19%) with 95% oxygen, ($p < 0.001$) (fig 1). During 40% supplemental oxygen therapy, only 11 patients had an SAOS of \leq 85% and none had an SAOS $<$ 80%. In

Abbreviations: CCHD, cyanotic congenital heart disease; SAOS, systemic arterial oxygen saturation

patients with a baseline SAOS < 85% we found a significant association between the SAOS response to supplementary oxygen and baseline haematocrit, consistent with a link between pulmonary vascular reactivity and the stimulus for erythropoiesis. Oxygen administration was associated with a significant decrease in heart rate (room air 78 (15.4) bpm, 40% oxygen 73 (11.4) bpm, and 95% oxygen 71 (10.9) bpm, $p < 0.001$), but no significant change in systemic systolic blood pressure (room air 115 (14) mm Hg, 40% oxygen 112 (15) mm Hg, and 95% oxygen 114 (17) mm Hg, $p =$ not significant).

DISCUSSION

The most likely mechanism of improvement in SAOS is pulmonary arterial vasodilation. Previously reported invasive experiments with 95–100% supplemental oxygen, in similar patient cohorts, confirmed that pulmonary vascular resistance decreased and pulmonary blood flow increased with little or no effect on right to left shunting.^{3–5} This high concentration of supplemental oxygen is not suitable for long term therapy, however, because it is associated with lung toxicity. However, 40% supplemental oxygen can be administered at home and is safe for long term use. If the saturation improvement seen in this study could be maintained in these patients by administering 40% supplemental oxygen at home, it may reduce erythrocytosis and have a beneficial impact on quality of life and level of symptoms. It is worthy of note that six of the 29 patients studied had a resting SAOS > 85% and none of these had a haematocrit > 0.65 or required venesection; this is in contrast to the

need for venesection in 11 of the 23 patients with an SAOS < 85%.

In light of the results of this small study, we suggest that the concept of home oxygen supplementation having no place in the therapy of adults with CCHD needs to be reconsidered.

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Inspiratory muscle load and capacity in chronic heart failure

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Background: Although breathlessness is common in chronic heart failure (CHF), the role of inspiratory muscle dysfunction remains unclear. We hypothesised that inspiratory muscle endurance, expressed as a function of endurance time (Tlim) adjusted for inspiratory muscle load and inspiratory muscle capacity, would be reduced in CHF.

Methods: Endurance was measured in 10 healthy controls and 10 patients with CHF using threshold loading at 40% maximal oesophageal pressure (Poes_{max}). Oesophageal pressure-time product (PTPoes per cycle) and Poes_{max} were used as indices of inspiratory muscle load and capacity, respectively.

Results: Although Poes_{max} was slightly less in the CHF group (−117.7 (23.6) v −100.0 (18.3) cm H₂O; 95% CI −37.5 to 2.2 cm H₂O, $p = 0.1$), Tlim was greatly reduced (1800 v 306 (190) s; 95% CI 1368 to 1620 s, $p < 0.0001$) and the observed PTPoes per cycle/Poes_{max} was increased (0.13 (0.05) v 0.21 (0.04); 95% CI −0.11 to −0.03, $p = 0.001$). Most of this increased inspiratory muscle load was due to a maladaptive breathing pattern, with a reduction in expiratory time (3.0 (5.8) v 1.1 (0.3) s; 95% CI 0.3 to 3.5 s, $p = 0.03$) accompanied by an increased inspiratory time relative to total respiratory cycle (Ti/Ttot) (0.43 (0.14) v 0.62 (0.07); 95% CI −0.3 to −0.1, $p = 0.001$). However, log Tlim, which incorporates the higher inspiratory muscle load to capacity ratio caused by this altered breathing pattern, was $\geq 85\%$ predicted in seven of 10 patients.

Conclusions: Although a marked reduction in endurance time was observed in CHF, much of this reduction was explained by the increased inspiratory muscle load to capacity ratio, suggesting that the major contributor to task failure was a maladaptive breathing pattern rather than impaired inspiratory muscle endurance.

▲ *Thorax* 2004;**59**:477–482.